WEST Search History

DATE: Wednesday, September 25, 2002

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=OR			
L8	opson$9 same atp same administ$9	0	L8
L7	antigen same atp same administ$9	7	L7
L6	antigen same atp same (rbc or ghost$4)	1	L6
L5	antigen same atp	415	L5
L4	antigen same atp same opson$9	0	L4
L3	L2 and atp	7	L3
L2	L1 and antigen	13	L2
L1	(silverstein)[in] or (loike) [in] or (divirgilia)[in]	400	L1

END OF SEARCH HISTORY

(FILE 'HOME' ENTERED AT 20:03:16 ON 25 SE[redacted]002)

FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 20:03:47 ON 25 SEP 2002
L1 1375 S SILVERSTEIN S?/AU OR LOIKE J?/AU OR DIVIRGILIO F?/AU
L2 304 S L1 AND IMMUN?
L3 11 S L1 AND (HISTOCOMPATIBILITY OR MHC)
L4 8 DUP REM L3 (3 DUPLICATES REMOVED)
L5 0 S (ANTGIEN (1N) PRESENT?) AND (HISTOCOMPATIBILITY OR MHC) AND (
L6 15 S ANTIGEN (P) ATP (P) PRESENT? (P) APC
L7 5 DUP REM L6 (10 DUPLICATES REMOVED)
L8 0 S OPSON? (P) GHOST? (P) ATP
L9 123 S OPSON? (P) ATP
L10 45 DUP REM L9 (78 DUPLICATES REMOVED)
L11 5 S L10 AND ANTIGEN

=> s l9 (P) administ?
L12 0 L9 (P) ADMINIST?

=> s l6 (P) administ?
L13 1 L6 (P) ADMINIST?

=> dis l13 ibib abs

L13 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2002:184925 CAPLUS
DOCUMENT NUMBER: 136:231233
TITLE: A method for inducing or suppressing immunity comprising phagocytic particles and ATP receptors
INVENTOR(S): Silverstein, Samuel C.; Loike, John D.; Divirgilio, Francesco
PATENT ASSIGNEE(S): The Trustees of Columbia University in the City of New York, USA
SOURCE: PCT Int. Appl., 74 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002020042	A1	20020314	WO 2001-US28171	20010907

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-658698 A 20000908

AB The invention provides methods of delivering an **antigen** to class I or class II MHC receptors to induce immunity against the **antigen** in a subject having a disease. This invention also provides methods of delivering an **antigen** to a class I or class II MHC receptor to suppress immunity against the **antigen** in a subject having a disease. The method comprises: (a) filling particles with the **antigen** and **ATP** resulting in Ag/**ATP**-filled particles; (b) coating the Ag/**ATP**-filled particles with a ligand for an **antigen-presenting** cell; (c) incubating the ligand-coated Ag/**ATP**-filled particles with ligand-binding **antigen-presenting** cells under conditions permitting the **APCs** to bind to the particles and **APC** phagolysosomes to ingest the ligand-coated Ag/**ATP**-filled particles to facilitate delivery of the ingested **antigen** to class I or class II MHC and expression on the surface of the **APC**; (d) incubating the Ag-**APCs** of step (c) with lymphocytes previously removed from the subject having the disease; and (e) **administering** the incubated lymphocytes of step (d) to the subject so as to induce or suppress immunity against the **antigen**. The **antigen-presenting** cells can also be **administered** to induce or suppress immunity against the **antigen**.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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 NEWS  9  Jun 03  New e-mail delivery for search results now available
 NEWS 10  Jun 10  MEDLINE Reload
 NEWS 11  Jun 10  PCTFULL has been reloaded
 NEWS 12  Jul 02  FOREGE no longer contains STANDARDS file segment
 NEWS 13  Jul 22  USAN to be reloaded July 28, 2002;
                  saved answer sets no longer valid
 NEWS 14  Jul 29  Enhanced polymer searching in REGISTRY
 NEWS 15  Jul 30  NETPIRST to be removed from STN
 NEWS 16  Aug 08  CANCERLIT reload
 NEWS 17  Aug 08  PHARMAMarketLetter(PHARMAML) - new on STN
 NEWS 18  Aug 08  NTIS has been reloaded and enhanced
 NEWS 19  Aug 19  Aquatic Toxicity Information Retrieval (AQUIRE)
                  now available on STN
 NEWS 20  Aug 19  IFIPAT, IFICDB, and IFIUDB have been reloaded
 NEWS 21  Aug 19  The MEDLINE file segment of TOXCENTER has been reloaded
 NEWS 22  Aug 26  Sequence searching in REGISTRY enhanced
 NEWS 23  Sep 03  JAPIO has been reloaded and enhanced
 NEWS 24  Sep 16  Experimental properties added to the REGISTRY file
 NEWS 25  Sep 16  Indexing added to some pre-1967 records in CA/CAPLUS
 NEWS 26  Sep 16  CA Section Thesaurus available in CAPLUS and CA

 NEWS EXPRESS  February 1 CURRENT WINDOWS VERSION IS V6.0d,
               CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP),
               AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
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FILE 'HOME' ENTERED AT 20:03:16 ON 25 SEP 2002

=> file medline caplus embase biosis
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21

FILE 'MEDLINE' ENTERED AT 20:03:47 ON 25 SEP 2002

FILE 'CAPLUS' ENTERED AT 20:03:47 ON 25 SEP 2002
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FILE 'BIOSIS' ENTERED AT 20:03:47 ON 25 SEP 2002
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=> s silverstein S?/au or loike J?/au or divirgilio F?/au
L1        1375 SILVERSTEIN S?/AU OR LOIKE J?/AU OR DIVIRGILIO F?/AU

=> s l1 and immun?
L2         304 L1 AND IMMUN?

=> s l1 and (histocompatibility or MHC)
L3          11 L1 AND (HISTOCOMPATIBILITY OR MHC)

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4            8 DUP REM L3 (3 DUPLICATES REMOVED)

=> dis l4 1-8 ibib abs

L4   ANSWER 1 OF 8  CAPLUS  COPYRIGHT 2002 ACS
ACCESSION NUMBER:        2002:184925  CAPLUS
DOCUMENT NUMBER:         136:231233
TITLE:                   A method for inducing or suppressing immunity
                         comprising phagocytic particles and ATP receptors
INVENTOR(S):             Silverstein, Samuel C.; Loike, John
                         D.; Divirgilio, Francesco
PATENT ASSIGNEE(S):      The Trustees of Columbia University in the City of New
                         York, USA
SOURCE:                  PCT Int. Appl., 74 pp.
                         CODEN: PIXXD2
```

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002020042	A1	20020314	WO 2001-US28171	20010907

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-658698 A 20000908

AB The invention provides methods of delivering an antigen to class I or class II **MHC** receptors to induce immunity against the antigen in a subject having a disease. This invention also provides methods of delivering an antigen to a class I or class II **MHC** receptor to suppress immunity against the antigen in a subject having a disease. The method comprises: (a) filling particles with the antigen and ATP resulting in Ag/ATP-filled particles; (b) coating the Ag/ATP-filled particles with a ligand for an antigen-presenting cell; (c) incubating the ligand-coated Ag/ATP-filled particles with ligand-binding antigen-presenting cells under conditions permitting the APCs to bind to the particles and APC phagolysosomes to ingest the ligand-coated Ag/ATP-filled particles to facilitate delivery of the ingested antigen to class I or class II **MHC** and expression on the surface of the APC; (d) incubating the Ag-APCs of step (c) with lymphocytes previously removed from the subject having the disease; and (e) administering the incubated lymphocytes of step (d) to the subject so as to induce or suppress immunity against the antigen. The antigen-presenting cells can also be administered to induce or suppress immunity against the antigen.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 2 OF 8 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 91037972 MEDLINE
DOCUMENT NUMBER: 91037972 PubMed ID: 2172452
TITLE: Murine cytotoxic T lymphocytes specific for herpes simplex virus type 1 recognize the immediate early protein ICP4 but not ICP0.
AUTHOR: Martin S; Zhu X X; **Silverstein S J**; Courtney R J; Yao F; Jenkins F J; Rouse B T
CORPORATE SOURCE: Department of Microbiology and Nutrition Research, Upjohn Company, Kalamazoo, Michigan 49001.
CONTRACT NUMBER: AI 14981 (NIAID)
CA 42460 (NCI)
GM 38125 (NIGMS)
+
SOURCE: JOURNAL OF GENERAL VIROLOGY, (1990 Oct) 71 (Pt 10) 2391-9. Journal code: 0077340. ISSN: 0022-1317.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199012
ENTRY DATE: Entered STN: 19910208
Last Updated on STN: 19980206
Entered Medline: 19901204

AB Vaccinia virus recombinants expressing the herpes simplex virus type 1 (HSV-1) genes encoding ICP0 or ICP4 were used to identify the precise target antigen(s) of murine anti-viral cytotoxic T lymphocytes (CTL) specific for the non-structural immediate early proteins. These studies revealed that HSV-1-specific CTL, restricted to class I major **histocompatibility** complex genes of the H-2k haplotype but not the H-2d or H-2b haplotypes, would lyse autologous cells expressing ICP4. HSV-1-specific CTL derived from various mice strains failed to lyse target cells expressing ICP0. Calculation of the frequencies of H-2k-restricted virus-specific CTL demonstrated that approximately a third of the total HSV-1-specific response was directed against ICP4. Immunization of mice with either recombinant vaccinia virus or transfected L cells expressing ICP4 induced HSV-1-specific lymphoproliferation and delayed hypersensitivity but CTLs were not induced. More importantly, such immunized animals were unable to resist or control a subsequent challenge with virulent HSV-1.

L4 ANSWER 3 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1988:450619 BIOSIS
DOCUMENT NUMBER: BR35:91499
TITLE: PROCESSING AND PRESENTATION OF ANTIGENS.
AUTHOR(S): PERNIS B; **SILVERSTEIN S C**; VOGEL H J
CORPORATE SOURCE: COLL. PHYS. SURG., COLUMBIA UNIV., NEW YORK, N.Y.
SOURCE: PERNIS, B., S. C. SILVERSTEIN AND H. J. VOGEL (ED.). PROCESSING AND PRESENTATION OF ANTIGENS. XIV+324P. ACADEMIC PRESS, INC.: SAN DIEGO, CALIFORNIA, USA; LONDON, ENGLAND, UK. ILLUS, (1988) 0 (0), XIV+342P.
ISBN: 0-12-551855-2.
DOCUMENT TYPE: Book
FILE SEGMENT: BR; OLD
LANGUAGE: English

AB Papers in this volume are the work of immunologists, biochemists, cell biologists, and virologists, and should be of interest to researchers in these disciplines. These papers on the early stages of the immune response are grouped under headings such as endosomes, lysosomes, and recycling, presentation in the context of class I major **histocompatibility** complex (**MHC**) molecules, and interactions of antigens with class II **MHC** molecules. The remaining parts deal with macrophages and dendritic cells as accessory cells, antigen presentation by B cells, and what T cells see. Illustrations and graphs supplement the text, and an index is provided.

L4 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1988:547499 CAPLUS
DOCUMENT NUMBER: 109:147499
TITLE: Localization of immune receptor recognition sites on major **histocompatibility** molecules through the analysis of H-2Kb mutants
AUTHOR(S): Zeff, Richard A.; Kumar, P. Ajit; Geliebter, Jan; Nathenson, Stanley A.

CORPORATE SOURCE: Dep. Cell Biol., Albert Einstein Coll. Med., Bronx, NY, 10461, USA
SOURCE: Process. Presentation Antigens (1988), 263-72. Editor(s): **Pernis, Benvenuto; Silverstein, Samuel C.; Vogel, Henry J.** Academic: San Diego, Calif. CODEN: 56HSAQ
DOCUMENT TYPE: Conference; General Review
LANGUAGE: English
AB A review with 33 refs. Studies on H-2Kb mutants suggest that interaction of these mols. with the T cell receptor app. occurs at sites that are formed from amino acid residues at localized positions on the .alpha.1 and .alpha.2 domains of the class I polypeptide.

L4 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1988:547498 CAPLUS
DOCUMENT NUMBER: 109:147498
TITLE: Recycling of **histocompatibility** molecules and antigen presentation
AUTHOR(S): Pernis, Benvenuto
CORPORATE SOURCE: Coll. Physicians Surg., Columbia Univ., New York, NY, 10032, USA
SOURCE: Process. Presentation Antigens (1988), 247-59. Editor(s): **Pernis, Benvenuto; Silverstein, Samuel C.; Vogel, Henry J.** Academic: San Diego, Calif. CODEN: 56HSAQ
DOCUMENT TYPE: Conference; General Review
LANGUAGE: English
AB A review with 44 refs. of the importance of the intracellular formation of complexes between immunogenic peptides and class I and class II **histocompatibility** antigens in antigen presentation to cytotoxic and helper T-lymphocytes.

L4 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1988:547495 CAPLUS
DOCUMENT NUMBER: 109:147495
TITLE: The role of **MHC** and amphipathic structures in T cell recognition: features determining immunodominance
AUTHOR(S): Berzofsky, Jay A.; Cease, Kemp B.; Berkower, Ira J.; Margalit, Hanah; Cornette, Jim; Spouge, John; Spencer, Cecilia; Buckenmeyer, Gail; Streicher, Howard; et al.
CORPORATE SOURCE: Natl. Cancer Inst., Natl. Inst. Health, Bethesda, MD, 20892, USA
SOURCE: Process. Presentation Antigens (1988), 125-31. Editor(s): **Pernis, Benvenuto; Silverstein, Samuel C.; Vogel, Henry J.** Academic: San Diego, Calif. CODEN: 56HSAQ
DOCUMENT TYPE: Conference; General Review
LANGUAGE: English
AB A review with 30 refs. of sperm whale myoglobin recognition by murine T cells. The immunodominance of the glutamyl residue 109 of the myoglobin is discussed with regard to its presence in an .alpha.-helix with amphipathic properties; amphipathic structures may be a prerequisite for antigen recognition by T cells. Dependence of T cell recognition of myoglobin on I-A and I-E class II **histocompatibility** antigens is also considered.

L4 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1988:627970 CAPLUS
DOCUMENT NUMBER: 109:227970
TITLE: The epitopes of influenza nucleoprotein recognized by cytotoxic T lymphocytes can be defined with short synthetic peptides
AUTHOR(S): Townsend, A. R. M.; Rothbard, J.; Gotch, F. M.; Bastin, J.; Bahadur, G.; Wraith, D.; McMichael, A. J.
CORPORATE SOURCE: Nuffield Dep. Clin. Med., John Radcliffe Hosp., Headington/Oxford, OX3 9DU, UK
SOURCE: Process. Presentation Antigens (1988), 81-5. Editor(s): **Pernis, Benvenuto; Silverstein, Samuel C.; Vogel, Henry J.** Academic: San Diego, Calif. CODEN: 56HSAQ
DOCUMENT TYPE: Conference; General Review
LANGUAGE: English
AB A review and discussion with 10 refs. of synthetic peptides that contain epitopes from influenza virus nucleoprotein and evidence that cytotoxic T-lymphocyte recognition of synthetic peptides is class I antigen-restricted.

L4 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1988:547493 CAPLUS
DOCUMENT NUMBER: 109:147493
TITLE: Pathways of viral antigen presentation in T lymphocyte recognition
AUTHOR(S): Braciale, T. J.; Morrison, L. A.; Henkel, T. J.; Braciale, V. L.
CORPORATE SOURCE: Sch. Med., Washington Univ., St. Louis, MO, 63110, USA
SOURCE: Process. Presentation Antigens (1988), 69-79. Editor(s): **Pernis, Benvenuto; Silverstein, Samuel C.; Vogel, Henry J.** Academic: San Diego, Calif. CODEN: 56HSAQ
DOCUMENT TYPE: Conference; General Review
LANGUAGE: English
AB A review with 22 refs. discussing evidence suggesting that class I and class II major **histocompatibility** complex-restricted T-lymphocytes recognize influenza antigens presented through distinctly different presentation pathways.

=> s (antgien (1N) present?) and (histocompatibility or MHC) and (red (1N) blood (1N) cell (1N) ghost)
3 FILES SEARCHED...
L5 0 (ANTGIEN (1N) PRESENT?) AND (HISTOCOMPATIBILITY OR MHC) AND (RED (1N) BLOOD (1N) CELL (1N) GHOST)

=> s antigen (P) ATP (P) present? (P) APC
L6 15 ANTIGEN (P) ATP (P) PRESENT? (P) APC

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7 5 DUP REM L6 (10 DUPLICATES REMOVED)

=> dis l7 1-5 ibib abs

L7 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2002:184925 CAPLUS
DOCUMENT NUMBER: 136:231233
TITLE: A method for inducing or suppressing immunity comprising phagocytic particles and ATP receptors
INVENTOR(S): Silverstein, Samuel C.; Loike, John D.; Divirgilio, Francesco
PATENT ASSIGNEE(S): The Trustees of Columbia University in the City of New York, USA
SOURCE: PCT Int. Appl., 74 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002020042	A1	20020314	WO 2001-US28171	20010907

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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-658698 A 20000908

AB The invention provides methods of delivering an **antigen** to class I or class II MHC receptors to induce immunity against the **antigen** in a subject having a disease. This invention also provides methods of delivering an **antigen** to a class I or class II MHC receptor to suppress immunity against the **antigen** in a subject having a disease. The method comprises: (a) filling particles with the **antigen** and **ATP** resulting in Ag/**ATP**-filled particles; (b) coating the Ag/**ATP**-filled particles with a ligand for an **antigen-presenting** cell; (c) incubating the ligand-coated Ag/**ATP**-filled particles with ligand-binding **antigen-presenting** cells under conditions permitting the **APCs** to bind to the particles and **APC** phagolysosomes to ingest the ligand-coated Ag/**ATP**-filled particles to facilitate delivery of the ingested **antigen** to class I or class II MHC and expression on the surface of the **APC**; (d) incubating the Ag-**APCs** of step (c) with lymphocytes previously removed from the subject having the disease; and (e) administering the incubated lymphocytes of step (d) to the subject so as to induce or suppress immunity against the **antigen**. The **antigen-presenting** cells can also be administered to induce or suppress immunity against the **antigen**.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 2 OF 5 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 97260241 MEDLINE
DOCUMENT NUMBER: 97260241 PubMed ID: 9106336
TITLE: Identification of two types of autoreactive T lymphocyte clones cultured from cardiac allograft-infiltrating cells incubated with recombinant mycobacterial heat shock protein 71.
AUTHOR: Liu K; Moliterno R A; Fu X F; Duquesnoy R J
CORPORATE SOURCE: Division of Transplantation Pathology, University of Pittsburgh Medical Center, PA 15261, USA.
CONTRACT NUMBER: AI-23567 (NIAID)
SOURCE: TRANSPLANT IMMUNOLOGY, (1997 Mar) 5 (1) 57-65.
Journal code: 9309923. ISSN: 0966-3274.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199706
ENTRY DATE: Entered STN: 19970620
Last Updated on STN: 19990129
Entered Medline: 19970612

AB Recent studies in several laboratories have advanced the concept that during cellular rejection, the allograft undergoes a stress response which regulates the expression of stress proteins (or heat shock proteins, hsp) and triggers the recruitment and activation of hsp-reactive lymphocytes. In a rat model of heterotopic heart transplants we have found that allograft-infiltrating lymphocytes respond to recombinant mycobacterial hsp and irradiated syngeneic spleen cells as a source of self-**APC** (**antigen-presenting** cells). This report describes T cell clones generated by culturing ACI into Lewis rat cardiac allograft-derived lymphocytes with mycobacterial hsp71, syngeneic spleen cells and IL-2 (interleukin-2). Two groups of self-**APC**-reactive T cell clones have been distinguished, all of them are CD3+, CD4+, CD8-. One group is referred to as hsp71-dependent, autoreactive T cells because these clones respond to self-**APC** but only in the presence of hsp71. No reactivity is seen with mycobacterial hsp65 or when hsp71 is tested with allo-PC from ACI donors or third-party **APC** from Brown Norway (BN) rats. Treatment of hsp71 with trypsin, polymyxin B or **ATP**-agarose chromatography abrogates the hsp71 effect thus indicating that structurally intact hsp71 must interact with self-**APC** which then activate hsp71-dependent, autoreactive T cells. The second group of clones reacts to self-**APC** and while their response does not require the presence of hsp71, their proliferation is often augmented by hsp71 but not by hsp65. These hsp71-independent, autoreactive clones do not respond to allo-**APC** from ACI donors or third-party **APC** from BN rats. Polymyxin or trypsin treatment had no significant effect on their proliferative responses. The data with the anti-TCR-alpha beta monoclonal antibody R73 offer additional evidence for two functionally different types of self-**APC** reactive CD4 cells infiltrating the allograft. R73 inhibits the proliferation of self-**APC** induced responses of hsp-71-independent clones as well as the allo-**APC** induced responses of alloreactive T cell clones. In contrast, this antibody augments the responses of hsp71-dependent T cells. Moreover, these clones can also proliferate in response to self-**APC** when hsp71 is substituted by R73. The hsp71-dependency of self-**APC** reactive T cell reactivity represents a previously unrecognized mechanism of cellular immunity to allografts. This mechanism might be related to the peptide binding properties of hsp71 and the ability of stress proteins to function as molecular chaperones in **antigen** processing.

L7 ANSWER 3 OF 5 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 95276250 MEDLINE
DOCUMENT NUMBER: 95276250 PubMed ID: 7538819
TITLE: Role of extracellular adenosine triphosphate in the cytotoxic T-lymphocyte-mediated lysis of antigen presenting cells.
AUTHOR: Blanchard D K; Wei S; Duan C; Pericle F; Diaz J I; Djeu J Y
CORPORATE SOURCE: Department of Medical Microbiology and Immunology, University of South Florida College of Medicine, Tampa, USA.
CONTRACT NUMBER: AI-33674 (NIAID)
SOURCE: BLOOD, (1995 Jun 1) 85 (11) 3173-82.
Journal code: 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199506
ENTRY DATE: Entered STN: 19950707
Last Updated on STN: 19970203
Entered Medline: 19950623

AB The lysis of **antigen presenting** cells (**APCs**) by cytotoxic T lymphocytes (CTLs) may be one mechanism whereby an immune response is downregulated by Staphylococcus superantigens. Disappearance of monocytes/macrophages from staphylococcal enterotoxin A (SEA)-activated peripheral blood mononuclear cell (PBMC) cultures, but not from control PBMC cultures was seen by flow cytometry. Recently, adenosine triphosphate (**ATP**) has been described as an effector molecule in CTL-mediated lysis of some murine tumor target cells. We have also shown that **ATP** caused the lysis of human macrophages, and that treatment of cells with interferon gamma (IFN gamma) rendered macrophages significantly more sensitive to **ATP** than untreated cells. To show that this purine nucleotide may play a role in modulating the immune system, we generated human CTLs that were stimulated with SEA, and used them as effector cells against SEA-pulsed autologous macrophages. CTLs were found to specifically lyse SEA-pulsed macrophages, while control, unpulsed, macrophages were unaffected. The addition of hexokinase, an enzyme that hydrolyzes **ATP**, significantly abrogated the killing of SEA-pulsed cells during the assay. In examining the mechanism of cytotoxicity, electron microscopy showed that macrophages incubated with both **ATP** and CTLs underwent necrosis, rather than apoptosis. From these results, it is suggested that **ATP** is released from CTLs during **antigen presentation**, and that IFN gamma-activated macrophages, which are inherently more sensitive to this mediator, are readily lysed and therefore removed from circulation, thus downregulating an immune response.

L7 ANSWER 4 OF 5 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 95394018 MEDLINE
DOCUMENT NUMBER: 95394018 PubMed ID: 7664779
TITLE: Establishment of a cell line with features of early dendritic cell precursors from fetal mouse skin.
AUTHOR: Girolomoni G; Lutz M B; Pastore S; Assmann C U; Cavani A; Ricciardi-Castagnoli P
CORPORATE SOURCE: Laboratory of Immunology, Istituto Dermopatico dell'Immacolata, IRCCS, Rome, Italy.
SOURCE: EUROPEAN JOURNAL OF IMMUNOLOGY, (1995 Aug) 25 (8) 2163-9.
Journal code: 1273201. ISSN: 0014-2980.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199510
ENTRY DATE: Entered STN: 19951020
Last Updated on STN: 19951020
Entered Medline: 19951010

AB During ontogeny, the skin is progressively populated by major histocompatibility complex class II-negative dendritic cell (DC) precursors that then mature into efficient **antigen-presenting** cells (**APC**). To characterize these DC progenitors better, we generated myeloid cell lines from fetal mouse skin by infecting cell suspensions with a retroviral vector carrying an envAKR-mycMH2 fusion gene. These cells, represented by the line FSDC, displayed a dendritic morphology and their proliferation in serum-free medium was promoted by granulocyte/macrophage colony-stimulating factor (GM-CSF), but not macrophage-CSF. FSDC expressed strong surface-membrane **ATP**/ADPase activity, intracellular staining for 2A1 **antigen**, and a surface phenotype consistent with a myeloid precursor: H-2d,b+, I-Ad,b+, CD54+, CD11b+, CD11c+, 2.4G2+, F4/80+, CD44+, 2F8+, ER-MP 12-, Sca-1+, Sca-2+, NLDC-145-, B7.2+, B7.1-, J11d-, B220-, Thy-1-, and CD3-. FSDC stimulated poorly allogeneic or syngeneic T cells in the primary mixed-leukocyte reaction, and markedly increased this function after treatment with GM-CSF, GM-CSF and interleukin (IL)-4 or interferon-gamma (IFN-gamma); in contrast, stem cell factor, IL-1 alpha and tumor necrosis factor-alpha had no effect. Preculture with IFN-gamma was required for **presentation** of haptens to primed T cells in vitro. However, FSDC, even after cytokine activation, were less potent **APC** than adult epidermal Langerhans cells in both of the above assays. Finally, FSDC derivatized with haptens and injected either intravenously or subcutaneously could efficiently induce contact sensitivity responses in naive syngeneic mice. The results indicate that fetal mouse skin is colonized by myeloid precursors possessing a macrophage/immature DC-like surface phenotype and priming capacity in vivo. These cells need further differentiation and activation signals (e.g. cytokines) to express their **antigen presenting** potential in vitro.

L7 ANSWER 5 OF 5 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 90063450 MEDLINE
DOCUMENT NUMBER: 90063450 PubMed ID: 2584924
TITLE: A peptide binding protein having a role in antigen presentation is a member of the HSP70 heat shock family.
AUTHOR: Vanbuskirk A; Crump B L; Margoliash E; Pierce S K
CORPORATE SOURCE: Department of Biochemistry, Molecular Biology and Cell Biology, Northwestern University, Evanston, Illinois 60208.
CONTRACT NUMBER: AI-12001 (NIAID)
AI-18939 (NIAID)
AI-23717 (NIAID)
SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (1989 Dec 1) 170 (6) 1799-809.
Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199001
ENTRY DATE: Entered STN: 19900328
Last Updated on STN: 19970203
Entered Medline: 19900108

AB The T cell recognition of globular protein **antigens** requires the processing and **presentation** of the **antigen** by Ia-expressing **APCs**. Processing is believed to involve the uptake of **antigen** into an acidic compartment where proteolysis occurs. The resulting peptides containing the T cell antigenic determinant are associated with Ia and **presented** at the cell surface to the specific T cells. The mechanisms by which antigenic peptides become associated with Ia is not known. We previously described a peptide binding protein of 72/74 x 10(3) Mr (PBP72/74) that plays a role in **antigen presentation** as shown by the ability of an antiserum raised in rabbits to affinity-purified PBP72/74 to block **presentation** of cytochrome c to a cytochrome c-specific T cell hybrid. Here we show that PBP72/74 is recognized by mAbs specific for members of the HSP70 family of proteins. In Western blots PBP72/74 is bound by mAb 7.10, specific for an evolutionarily conserved epitope of HSP proteins and by mAb N27, specific for both the constitutively expressed and inducible 72/73 x 10(3) Mr HSP70 proteins. In addition, PBP72/74 shares a second common feature of the HSP proteins, that of binding to **ATP**. Indeed, **ATP** causes the release of PBP72/74 from binding to a peptide fragment of cytochrome c (Pc 81-104) and PBP72/74 can be eluted from **ATP** columns by Pc 81-104. Finally, a portion of PBP72/74 is shown to be **present** on B cell surfaces by immunofluorescence staining. Thus, it appears that characteristics of the heat shock proteins are shared by a protein playing a role in **antigen presentation**, suggesting some commonality in function.

=> s opson? (P) ghost? (P) ATP
L8 0 OPSON? (P) GHOST? (P) ATP

=> s opson? (P) ATP
L9 123 OPSON? (P) ATP

=> dup rem l9
PROCESSING COMPLETED FOR L9
L10 45 DUP REM L9 (78 DUPLICATES REMOVED)

=> s l10 and antigen
L11 5 L10 AND ANTIGEN

=> dis l11 1-5 ibib abs

L11 ANSWER 1 OF 5 MEDLINE
ACCESSION NUMBER: 95359458 MEDLINE
DOCUMENT NUMBER: 95359458 PubMed ID: 7632965
TITLE: Influence of glutamine on the phenotype and function of human monocytes.
AUTHOR: Spittler A; Winkler S; Gotzinger P; Oehler R; Willheim M; Tempfer C; Weigel G; Fugger R; Boltz-Nitulescu G; Roth E
CORPORATE SOURCE: Department of Surgery, University of Vienna, Austria.
SOURCE: BLOOD, (1995 Aug 15) 86 (4) 1564-9.
Journal code: 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199509
ENTRY DATE: Entered STN: 19950921
Last Updated on STN: 19980206
Entered Medline: 19950914

AB Reduced concentrations of glutamine (GLN) in plasma and skeletal muscle, defective host defense systems, and a diminished expression of the HLA-DR **antigen** on monocytes are important diagnostic parameters for late post-injury sepsis. In this in vitro study, we investigated whether blood monocyte-derived macrophage **antigen** expression and function from healthy donors is influenced by GLN. Lowering the GLN concentration in culture medium from 2 mmol/L to 200 mumol/L reduced the expression of HLA-DR by 40% (P < .001) on monocyte-derived macrophages, and decreased tetanus toxoid-induced **antigen** presentation. In addition, low GLN levels downregulated the expression of intercellular adhesion molecule-1 (ICAM-1/CD54), Fc receptor for IgG (Fc gamma RI/CD64), and complement receptors type 3 (CR3; CD11b/CD18) and type 4 (CR4; CD11c/CD18). A correlation was found between the phagocytosis of IgG-sensitized ox erythrocytes or **opsonized** Escherichia coli and the decreased expression of Fc gamma RI and CR3. Monocyte expression of CD14, CD71, and Fc gamma RIII/CD16 and capacity to phagocytose latex beads were not affected by altering the level of GLN. Depletion of GLN was associated with a significant reduction in cellular adenosine triphosphate (**ATP**), which may have influenced cell surface marker expression and phagocytosis. It remains to be seen whether these in vitro findings are of clinical significance in the treatment of sepsis.

L11 ANSWER 2 OF 5 MEDLINE
ACCESSION NUMBER: 90234863 MEDLINE
DOCUMENT NUMBER: 90234863 PubMed ID: 2184903
TITLE: Regulation of autoimmune anti-platelet antibody-mediated adhesion of monocytes to platelet GPIIb/GPIIIa: effect of armed monocytes and the Mac-1 receptor.
AUTHOR: Hymes K B; Schuck M P; Karpatkin S
CORPORATE SOURCE: New York University Medical School, NY 10016.
CONTRACT NUMBER: HL-13336-17 (NHLBI)
HL01821 (NHLBI)
SOURCE: BLOOD, (1990 May 1) 75 (9) 1813-9.
Journal code: 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199006
ENTRY DATE: Entered STN: 19900706
Last Updated on STN: 19900706
Entered Medline: 19900606

AB Platelet autoantigen-autoantibody-monocyte interaction was studied by utilization of a specific monoclonal antibody (MoAb) 10E5 to trap and immobilize the GPIIb-GPIIIa complex on microtiter plates. Peripheral blood mononuclear cells (PBMC) or purified monocytes formed distinct morphologic

clusters after incubation with immobilized **antigen** for 18 hours at 37 degrees C. PBMC of 18 and 19 patients with autoimmune thrombocytopenic purpura (**ATP**) formed 48 +/- 6.8 (SEM) clusters/well compared with 7.4 +/- 1.0 for control subjects, P less than .001. The number of clusters per well correlated inversely and exponentially with platelet count, r = -.8, n = 21, indicating that the GPIIb-GPIIIa autoantigen is pathophysiologically relevant. Binding of **ATP** PBMC to immobilized GPIIb-GPIIIa could be inhibited by F(ab')2 fragments of immunoglobulin (Ig) G of **ATP** patients, indicating that monocyte IgG bound to autoantigen by its F(ab')2 domain. Optimal cluster formation could be obtained with normal monocytes if preincubated with **ATP** IgG but not with F(ab')2 fragments of **ATP** IgG, indicating that **ATP** IgG binds to monocytes by its Fc domain. Armed monocytes (ie, normal monocytes preincubated with **ATP** IgG) bound to immobilized autoantigen 5.8-fold greater than normal monocytes incubated with immobilized autoantigen **opsonized** with **ATP** IgG. Armed monocyte adhesion could be inhibited 81% from 18.9 +/- 1.6 to 3.6 +/- 0.5 clusters/well by prior fixation with 0.1% formalin, whereas fixation of IgG before arming of monocytes was not inhibitory. MoAb MM41, directed against the alpha m-chain of the Mac-1 adhesive protein receptor of monocytes, inhibited cluster formation by 79%. Thus, (1) armed monocyte interaction with autoantigen is considerably more effective than monocyte interaction with **opsonized** autoantigen; (2) armed monocyte interaction requires specific F(ab')2-**antigen** recognition; and (3) monocyte-autoantigen interaction requires a secondary nonimmunologic adhesive event.

L11 ANSWER 3 OF 5 MEDLINE
ACCESSION NUMBER: 83304098 MEDLINE
DOCUMENT NUMBER: 83304098 PubMed ID: 6604367
TITLE: [Cellular and humoral immune phenomena in psoriatic arthritis].
Untersuchungen uber zellulare und humorale Immunphanomene bei der Arthritis psoriatica.
AUTHOR: Neumuller J; Senautka G; Dunky A; Neumann H; Mayer F; Much T; Eberl R; Partsch G
SOURCE: WIENER KLINISCHE WOCHENSCHRIFT, (1983 Jun 10) 95 (12) 416-22.
Journal code: 21620870R. ISSN: 0043-5325.
PUB. COUNTRY: Austria
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198310
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19900319
Entered Medline: 19831021

AB The function of cellular immunity factors (lymphocyte transformation and phagocytosis by polymorphonuclear leucocytes [PMN] and monocytes in connection with the concentration of intracellular **ATP**) and humoral immunity factors (serum concentration of immunoglobulin and complement factors C'3 and C'4) was investigated in 16 controls, 21 patients with psoriatic arthritis and 19 with psoriasis vulgaris. The results were compared with the clinical and anamnestic data of the patients. PMN phagocytosis of zymosan **opsonized** with rabbit standard serum was decreased in psoriasis vulgaris in comparison with the controls. Also, monocyte phagocytosis of non-**opsonized** zymosan was decreased in psoriatic arthritis, as compared with psoriasis vulgaris. Furthermore, in PMNs intracellular **ATP** was elevated in psoriatic arthritis as compared with the controls, but decreased in comparison with patients with psoriasis vulgaris. The intracellular **ATP** in monocytes was decreased in psoriasis vulgaris as compared with the controls. Humoral immunological findings: serum IgG concentration was higher in psoriatic arthritis than in controls and in psoriasis vulgaris. Elevated C'3 and decreased C'4 serum concentrations in psoriatic arthritis indicate an activation of the complement system.

L11 ANSWER 4 OF 5 MEDLINE
ACCESSION NUMBER: 83153661 MEDLINE
DOCUMENT NUMBER: 83153661 PubMed ID: 6830791
TITLE: Binding of autologous IgG to human red blood cells before and after ATP-depletion. Selective exposure of binding sites (autoantigens) on spectrin-free vesicles.
AUTHOR: Muller H; Lutz H U
SOURCE: BIOCHIMICA ET BIOPHYSICA ACTA, (1983 Apr 6) 729 (2) 249-57.
Journal code: 0217513. ISSN: 0006-3002.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198305
ENTRY DATE: Entered STN: 19900318
Last Updated on STN: 19900318
Entered Medline: 19830527

AB Binding of autologous IgG to fresh, **ATP**-depleted red blood cells as well as to spectrin-free vesicles was studied by a non-equilibrium binding assay using 125I-iodinated protein A from Staphylococcus aureus. IgG binding was 14-times higher to spectrin-free vesicles than to **ATP**-maintaining red blood cells and 4-times higher than to **ATP**-depleted erythrocytes from which these vesicles were released. Protein A binding to vesicles that were released from washed and nutrient-deprived erythrocytes, was dependent on added autologous IgG. However, spectrin-free vesicles that were spontaneously released from erythrocytes conserved in whole blood, bound similar amounts of protein A with or without added autologous IgG (0.45-0.55 ng/micrograms band 3 protein). These findings demonstrate that **opsonization** of spectrin-free vesicles by autologous IgG occurs not only in the test tube, but also under blood blank conditions. The binding characteristics of IgG to spectrin-free vesicles are indicative of a natural autoantibody rather than an unspecific binding of autologous IgG. The preferential binding of IgG to spectrin-free vesicles implies a selective exposure of corresponding autoantigens in membrane regions that have lost cytoskeletal anchorage and bud off.

L11 ANSWER 5 OF 5 MEDLINE
ACCESSION NUMBER: 81000919 MEDLINE
DOCUMENT NUMBER: 81000919 PubMed ID: 6157441
TITLE: Autoimmune thrombocytopenic purpura.
AUTHOR: Karpatkin S
SOURCE: BLOOD, (1980 Sep) 56 (3) 329-43. Ref: 157
Journal code: 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198011
ENTRY DATE: Entered STN: 19900316
Last Updated on STN: 19970203
Entered Medline: 19801125

AB Adult autoimmune thrombocytopenic purpura (**ATP**) is a platelet disorder that develops in certain individuals with a genetic as well as sex (female) predisposition following an environment event (?viral). This results in the production of an IgG antiplatelet antibody capable of reacting with the host's platelets, as well as crossing the placenta. This leads to the rapid clearance and destruction of **opsonized** platelets by the reticuloendothelial system, particularly the spleen, by greater than tenfold the normal rate. Bound platelet IgG correlates with disease severity, whereas serum antiplatelet IgG does not. It has not been rigorously established whether bound platelet IgG is directed against a platelet **antigen** or represents an immune complex bound to the platelet Fc receptor. Nevertheless, several lines of evidence suggest that antiplatelet IgG binds directly to a platelet **antigen**(s). Megakaryocyte number, volume, and mass are increased commensurate with increased platelet turnover. Platelets of increased size, megathrombocytes, are noted on peripheral smear or via platelet volume distribution analysis. Megathrombocyte number is proporationate to megakarocyte number and to platelet turnover. Megathrombocyte diameter is inversely proportional to platelet survival. Antiplatelet antibody is also associated with qualitative platelet functional defects, which are indistinguishable from those noted with thrombopathia (i.e., apparent platelet release defect). Antibody-induced functional defects are probably more common than quantitative thrombocytopenic defects and may represent a significant portion of those women with the "easy bruising" syndrome and normal platelet count. Adults who develop **ATP** generally develop the chronic variety, which remains permanently with the patient. Treatment should be directed towards maintaining the patient free of purpura, not restoring the platelet count to normal. This can generally be accomplished with a platelet count of > 40,000/cu mm with patients having this disorder. Approximately 50% of patients respond to steroids by a significant elevation of platelet count and improvement of purpura. However, cessation of therapy results in eventual relapse if the disease is of the chronic variety. Splenectomy is successful in approximately 65-75% of patients, resulting in a restoration of the platelet count to normal or safe levels by removing a major source of platelet destruction as well as antibody production; platelet survival improves. At least 50% of patients "in remission" following steroids or splenectomy generally have a compensated thrombocytolytic state in which increased platelet production keeps up with increased platelet destruction. Antiplatelet IgG can often be found in the serum of these patients. Patients refractory to steroids and/or splenectomy present with a serious therapeutic problem. Immunosuppressive therapy is effective in approximately one-third of refractory patients, but often relapses occur, requiring maintenance therapy with potentially mutagenic drugs...

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=> dis his

     (FILE 'HOME' ENTERED AT 20:03:16 ON 25 SEP 2002)

     FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 20:03:47 ON 25 SEP 2002
L1          1375 S SILVERSTEIN S?/AU OR LOIKE J?/AU OR DIVIRGILIO F?/AU
L2           304 S L1 AND IMMUN?
L3            11 S L1 AND (HISTOCOMPATIBILITY OR MHC)
L4             8 DUP REM L3 (3 DUPLICATES REMOVED)
L5             0 S (ANTGIEN (1N) PRESENT?) AND (HISTOCOMPATIBILITY OR MHC) AND (
L6            15 S ANTIGEN (P) ATP (P) PRESENT? (P) APC
L7             5 DUP REM L6 (10 DUPLICATES REMOVED)
L8             0 S OPSON? (P) GHOST? (P) ATP
L9           123 S OPSON? (P) ATP
L10           45 DUP REM L9 (78 DUPLICATES REMOVED)
L11            5 S L10 AND ANTIGEN

=> s l9 (P) administ?
L12            0 L9 (P) ADMINIST?

=> s l6 (P) administ?
L13            1 L6 (P) ADMINIST?

=> dis l13 ibib abs

L13  ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2002 ACS
ACCESSION NUMBER:         2002:184925  CAPLUS
DOCUMENT NUMBER:          136:231233
TITLE:                    A method for inducing or suppressing immunity
                          comprising phagocytic particles and ATP receptors
INVENTOR(S):              Silverstein, Samuel C.; Loike, John D.; Divirgilio,
                          Francesco
PATENT ASSIGNEE(S):       The Trustees of Columbia University in the City of New
                          York, USA
SOURCE:                   PCT Int. Appl., 74 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     --------------    ----  --------       ---------------  --------
     WO 2002020042      A1   20020314       WO 2001-US28171  20010907
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL,
             PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG,
             UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
             DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
             BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.:                    US 2000-658698   A  20000908
AB   The invention provides methods of delivering an antigen to class
     I or class II MHC receptors to induce immunity against the antigen
     in a subject having a disease.  This invention also provides methods of
     delivering an antigen to a class I or class II MHC receptor to
     suppress immunity against the antigen in a subject having a
     disease.  The method comprises: (a) filling particles with the
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antigen and **ATP** resulting in Ag/**ATP**-filled particles; (b) coating the Ag/**ATP**-filled particles with a ligand for an **antigen-presenting** cell; (c) incubating the ligand-coated Ag/**ATP**-filled particles with ligand-binding **antigen-presenting** cells under conditions permitting the **APCs** to bind to the particles and **APC** phagolysosomes to ingest the ligand-coated Ag/**ATP**-filled particles to facilitate delivery of the ingested **antigen** to class I or class II MHC and expression on the surface of the **APC**; (d) incubating the Ag-**APCs** of step (c) with lymphocytes previously removed from the subject having the disease; and (e) **administering** the incubated lymphocytes of step (d) to the subject so as to induce or suppress immunity against the **antigen**. The **antigen-presenting** cells can also be **administered** to induce or suppress immunity against the **antigen**.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT